isolate the patient or to examine the contacts. Because the latter look healthy they are ignored; disastrous consequences result from failure to trace the source of the infection. A coughing grandmother may be responsible, but because of the widespread impression that old people have an immunity to tuberculosis, the source is overlooked. Even if the diagnosed patient is isolated, the danger to the other members of the family still exists. The responsibility of examining the entire household rests with the attending physician. We have often found that innumerable patients have been diagnosed as tuberculous for years, but that the physicians had neglected to report them to the local department of public health, and that the contacts had never been examined.

Space prevents further discussion of mistakes in treatment. Osler epitomized the importance of diagnosis and treatment in these memorable words: "The ordinary physician treats the patient with medicine and without rest—rest, fresh air, and proper diet, the three things which will arrest a large number of cases of pulmonary tuberculosis *if,* and the if is *you,* gentlemen, the diagnosis is made early."

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REGULATION OF THE BLOOD SUGAR

The prevailing conception of the regulation of blood sugar, as exemplified by the events taking place after a meal or a dextrose tolerance test, may be summarized as follows:

1. The absorption of carbohydrates stimulates the pancreas to an increased output of insulin.

2. The increase in insulin causes greater storage and oxidation of sugar.

3. Excessive postprandial hyperglycemia in diabetes mellitus is due to failure of the pancreas to secrete increased amounts of insulin in response to absorbed carbohydrates.

4. The "diabetic" type of curve obtained after a dextrose tolerance test in starvation is due to lack of the proper response from the pancreas, which needs frequent stimulation in order to respond normally. Conversely, repeated or prolonged administration of dextrose results in greater tolerance for sugar due to increased mobilization of insulin in response to the previously administered sugar.

In 1932 Althausen and Thoenes,[1] in studying the influence of hepatic toxins on carbohydrate metabolism, found that increasing injury to the liver caused progressive impairment in dextrose tolerance. On the other hand, hyperactivity of the liver during periods of recovery with active regeneration of hepatic parenchyma, was observed to produce greater than normal tolerance to dextrose. These experiments showed that in the presence of an intact pancreas the liver is the organ which determines the shape of the dextrose tolerance curve.

These findings were confirmed and extended by a group of Chicago workers under the leadership of Soskin, who showed that the pancreas is not essential to the metabolic reactions which determine the normal dextrose tolerance curve. Soskin et al.[2,3,4] obtained normal dextrose tolerance curves (including adjustment to prolonged administrations of dextrose) in pancreatectomized dogs, in which a constant injection of insulin was substituted for the pancreas. In this way, the animals received an adequate supply of insulin to maintain a constant blood sugar, but were unable to mobilize additional insulin when sugar was given. If increased secretion of insulin were essential for a normal dextrose tolerance curve, these animals should have had abnormal curves, which was not the case. In another set of experiments, the same workers found normal dextrose tolerance curves in depancreatized and hypophysectomized dogs which had received no insulin for weeks.

These researches led Soskin to a new theory of blood-sugar regulation, in which the emphasis is shifted from the pancreas to the liver. According to this theory, the liver, in response to administered dextrose, decreases its output of sugar into the blood. However, the liver can respond properly to administration of sugar only under the influence of a suitable endocrine balance which, for simplicity, may be considered to consist of the opposing influences of the hormones of the pancreas and of the hypophysis. When this endocrine balance is disturbed by insufficiency of insulin or excessive activity of the hypophysis, the liver is not stimulated to inhibit its release of sugar until the blood sugar may greatly exceed the normal level, and the clinical picture of diabetes mellitus is produced. One might add that the reverse is also true, namely, when the endocrine balance is disturbed by excess of insulin or insufficient activity of the hypophysis, the clinical picture of "hyperinsulinism" is the result.

In conclusion, it must be said that the experiments just discussed do not disprove an increase in the secretion of insulin under the stimulus of a rise in blood sugar, for which there is evidence from experiments with transplanted pancreatic tissue. Also, the new conception of the regulation of blood sugar has as yet no practical application in the treatment of diabetes mellitus, but it does furnish an adequate explanation for the failure of partial pancreatectomy to cure some cases of so-called "hyperinsulinism," in which no tumor of the pancreas is present.

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